Mycobacterial Diseases
ISSN: 2161-1068
OPEN ACCESS Freely available online
Perspective

Drugs Involved in the Treatment of Mycobacterial Diseases

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DESCRIPTION

Mycobacteria are a group of bacteria that includes several species, such as *Mycobacterium tuberculosis*, the causative agent of tuberculosis, and *Mycobacterium leprae*, the causative agent of leprosy. These bacteria are difficult to treat due to their unique cell wall structure, which is rich in lipids and mycolic acids, and their ability to persist within host cells. The mycobacterial pharmacology is a complex field that requires a multidisciplinary approach. Antimicrobial therapy is the cornerstone of mycobacterial pharmacology. The first-line drugs used to treat tuberculosis include isoniazid, rifampin, ethambutol, and pyrazinamide. These drugs are typically used in combination, known as directly Observed Therapy Short-Course (DOTS), to prevent the development of drug resistance and improve treatment outcomes. Second-line drugs, such as fluoroquinolones, aminoglycosides, and linezolid, may be used when the first-line drugs are ineffective or the strain of tuberculosis is resistant to them.

Isoniazid is a first-line drug that inhibits the synthesis of mycolic acids, which are essential components of the mycobacterial cell wall. It is a prodrug that is activated by the mycobacterial enzyme KatG. Resistance to isoniazid is commonly associated with mutations in the *katG* gene or inactivating mutations in the *inhA* gene, which encodes an enzyme involved in mycolic acid synthesis. Isoniazid is well-tolerated, but it can cause hepatotoxicity, especially in patients with underlying liver disease. Rifampin is another first-line drug that inhibits the synthesis of RNA by binding to the beta subunit of the bacterial RNA polymerase. Resistance to rifampin is commonly associated with mutations in the *rpoB* gene, which encodes the beta subunit of RNA polymerase. Rifampin is highly effective in the treatment of tuberculosis, but it can cause hepatotoxicity and drug interactions due to its ability to induce cytochrome P450 enzymes. Ethambutol is a first-line drug that inhibits the synthesis of arabinogalactan, another component of the mycobacterial cell wall. It is usually given in combination with isoniazid, rifampin, and pyrazinamide. Resistance to ethambutol is uncommon, but it can cause optic neuritis, especially in patients with renal impairment. Pyrazinamide is a first-line drug that is believed to inhibit the synthesis of mycolic acids by lowering the intracellular pH of mycobacteria. Resistance to pyrazinamide is uncommon, but it can cause hepatotoxicity and hyperuricemia.

Second-line drugs are typically used in combination with first-line drugs when the latter are ineffective or the strain of tuberculosis is resistant to them. Fluoroquinolones, such as levofloxacin and moxifloxacin, are synthetic antibiotics that inhibit DNA gyrase and topoisomerase IV, enzymes involved in DNA replication and repair. Aminoglycosides, such as amikacin and kanamycin, are bactericidal antibiotics that bind to the 30S subunit of the bacterial ribosome, thereby inhibiting protein synthesis. Linezolid is an oxazolidinone antibiotic that inhibits protein synthesis by binding to the 50S subunit of the bacterial ribosome. The treatment of leprosy also requires a multidrug regimen. The first-line drugs used to treat leprosy include dapsone, rifampin, and clofazimine. Dapsone is a bacteriostatic drug that inhibits the synthesis of dihydrofolic acid.

CONCLUSION

Mycobacterial pharmacology is a complex and evolving field that is focused on the development and optimization of drugs to treat infections caused by mycobacteria, including the notorious *Mycobacterium tuberculosis*. The treatment of these infections is complicated by several factors, including the slow growth rate of mycobacteria, their ability to enter a dormant state within the host, and the emergence of drug-resistant strains. Nevertheless, significant progress has been made in recent years, and there are now several effective drugs available for the treatment of mycobacterial infections. These drugs act through various mechanisms, including inhibition of cell wall synthesis, interference with DNA replication, and disruption of protein synthesis.

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Received: 01-Feb-2023, Manuscript No. MDTL-23-23096; **Editor assigned:** 03-Feb-2023, Pre QC No. MDTL-22-23096 (PQ); **Reviewed:** 17-Feb-2023, QC No. MDTL-23- 23096; **Revised:** 24-Feb-2023, Manuscript No. MDTL-23-23096 (R); **Published:** 03-Mar-2023, DOI: 10.35248/2161-1068.23.13.326.

Citation: Smith C (2023) Drugs Involved in the Treatment of Mycobacterial Diseases. Mycobact Dis. 13:326.